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PATHWAYS AND SIGNIFICANCE OF REDISTRIBUTION OF ZINC IN PATIENTS WITH CHRONIC PANCREATITUS

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(O putyakh i zhachenii pereraspredeleniya tsinka u bol'nykh khronicheskim pankreatitom)

N. A. Zhukov and T. S. Bakhina

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PATHWAYS AND SIGNIFICANCE OF REDISTRIBUTION OF ZINC IN PATIENTS WITH CHRONIC PANCREATITIS

N. A. Zhukov and T. S. Bakhina*

Results are given of investigation of zinc by the method of chemico-spectral analysis in 100 patients with chronic pancreatitis and 50 healthy individuals. In patients with chronic pancreatitis there was noted a significant increase of the content of zinc in the blood at the expense of the increase of its concentration in erythrocytes and decrease in the blood plasma. On the basis of experimental investigations the authors regard redistribution of zinc under the conditions of affection of the insular apparatus in the pancreas as a compensatory phenomenon.

Since the publication of Okamoto's work, it has been known that the islands of Langerhans in the pancreas (β- and α-cells) contain a large quantity of zinc. It has been suggested that the zinc, binding with insulin, deposits the latter in the granules of the β-cells. An increase in blood sugar, by stimulating the excretion of insulin is accompanied by a rapid and sometime complete disappearance of zinc

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from them (Maske). Upon normalization of the blood sugar level, the zinc is again stored in the β-cells. Hence, this trace element migrates from the islands of Langerhans and back again under physiological conditions. Such mobility may occur only in the event of a rapid exchange of zinc between the cells in the islands of the pancreas and the blood. A study of the zinc content of the blood, which we performed using the method of chemico-spectral analysis in 50 healthy individuals, indicated that zinc is contained in both the blood plasma and the erythrocytes. Its concentration in both the whole blood and its component parts, according to our data, does not vary from the results obtained by other authors.

By studying the zinc content in the blood of 100 patients with chronic pancreatitis, we found that it showed a reliable increase in comparison with healthy individuals; this rise occurred only due to a rise in its concentration in the erythrocytes (the data are statistically reliable) with a simultaneous reliable decrease of the zinc level in the plasma. Thus, our data indicate that patients with chronic pancreatitis undergo a redistribution of zinc between the plasma and the erythrocytes. However, if this were the only pathway for increase of zinc content in the erythrocytes, on the whole its level in the blood would not change. Consequently, there is another way for the zinc to reach the erythrocytes — from the tissues of the organism. If we consider that patients with chronic pancreatitis frequently develop insular insufficiency with disruption of carbohydrate metabolism (we observed it in 21%), i.e., conditions are created in which, as we know, zinc can leave the β-cells, we must assume that its increased content in the erythrocytes can occur as the result of migration from the pancreas.

How are we to explain the concentration of zinc in the erythrocytes of patients with chronic pancreatitis with a decrease of this metal in the plasma? Considering the regulatory role of blood sugar in the exchange of zinc between the β-cells of the pancreas and the blood, we decided to study the influence of zinc in the erythrocytes on carbohydrate metabolism. We assumed that the zinc in the

erythrocytes cannot be linked to either insulin or glucagon since, as Williams' data indicate, both hormones, labelled with I^{131}, are not absorbed by erythrocytes.

We collected blood twice for the study, from the veins of three patients with chronic pancreatitis — fasting, and 60 minutes after taking 60 grams of glucose internally. The carefully washed erythrocytes were hemolyzed with distilled water and the hemolysate was used for the classic London test on 18 white rats. All of the rats showed a pronounced rise in blood sugar. Since Weitzel et al. indicate that zinc has a hyperglycemic effect, there is a basis for suggesting that the hyperglycemic effect in our tests was produced by the zinc. We shall consider the mechanism of this hyperglycemia from the standpoint of the ability of the zinc at the pH of the blood to form gelatinous complexes with insulin, which lower the solubility of the latter. This leads to incipient temporary hypoinsulinism and accompanying hyperglycemia.

On the basis of experimental studies, we can conclude that the redistribution of zinc with an increase in its content in the erythrocytes and the decrease in the plasma reduces the possibility of hyperglycemia and may be viewed under conditions of irritation of the pancreas and hypoinsulinism as a compensatory occurrence. This is supported by the fact that in our experiments on 30 patients with chronic pancreatitis a relationship was found between the effectiveness of the treatment and the zinc level in the plasma. As the condition of the patients improves under the influence of treatment, it is primarily the level of zinc in the plasma that begins to rise.